muscular relaxation or hypotonia of locomotor ataxia in the lower extremities, together with other symptoms of both diseases. A sister that had died was said to have presented a similar symptom complex. L. Bruns[12] reports the case of a man, aged sixty-four years, exhibiting a combination of symptoms of both locomotor ataxia and paralysis agitans. H. Eichhorst[13] reports a fatal case of locomotor ataxia in a man in whom the tremor of paralysis agitans developed a short while before death. Postmortem examination disclosed the anatomical lesions of locomotor ataxia.

Conclusions. From the foregoing facts and considerations it may be concluded that locomotor ataxia and paralysis agitans have little or nothing in common, etiologically, anatomico-pathologically, or symptomatologically. Their associated occurrence in a given case, therefore, must be looked upon as a mere coincidence. While by no means rare, neither disease is common, and their association is correspondingly infrequent. Only a small number of cases have been placed on record in which both diseases were present in the same patient.

A CASE OF CEREBRAL TUMOR PRESENTING A VERY UNUSUAL CLINICAL COURSE.[1]

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The following case seems to be one of unusual interest from the very peculiar, almost rhythmical, course that it pursued. Mrs. S., aged forty-six years, a married woman, and the mother of three healthy children, was first seen in May, 1906, complaining of severe headaches and constipation. The headaches had occurred during most of her adult life, and were nearly always associated with the menstrual period. While they were on she was scarcely able to speak, and often was sick at the stomach. Except for these headaches and the chronic constipation, she was and always had been well and full of healthy activity.

Her father died at an advanced age of cerebral hemorrhage; her mother at seventy years of a protracted diarrhœa. One brother died of angina pectoris, and was found to have marked sclerosis of the coronary and cerebral arteries.

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[12] Neurolog. Centralbl., 1904, xxiii, 978.

[13] Pathologie und Therapie der Nervenkrankheiten, Berlin and Vienna, 1907, p. 340.

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 11 and 12, 1909.

At the first consultation, in May, 1906 (about two years before her death), she was a healthy looking woman, and, as stated above, only complained of headaches and of chronic constipation. She was given a palliative and a cascara mixture to take regularly.

On June 6, 1906, it was noted that she looked "run down," spoke in a scanning manner, and wrote with difficulty, often leaving out words. The headaches were less severe. She was sent away for a long change. In October she was "looking fairly well," and had had no headache for a month. Bowels were regular with the cascara mixture. On November 6, for some days she had had occasional numbness and tingling in the left arm and hand. Right grip was sixty, and left fifty. There was no real numbness. The knee-jerks were normal. On December 10 it was noted that the patient had slight headache and was feeling dull, had difficulty in finding the right words, and "often says and writes wrong ones." Bowels were regular with medicine, and all other functions seemed to be normal. There had been no menstruation since last May. On May 15, 1907, the following note was made: "She is feeling depressed and easily worried, and has frequent headaches. Her weight keeps up. The hemoglobin registers 80 per cent. There has been no menstruation since last May."

She went away for a long change, and on returning the following note was made: "September 20, 1907.—Back from a long holiday, and looks fairly well, but faded. Hemoglobin, 80 per cent. If her bowels do not move she has severe headaches. Still has some hesitation in speech, and tends to use wrong words. Scarcely ever reads, and seldom writes letters, and if she does, may write wrong words. Sleeps well, and digestion is normal. The knee-jerks are equal and sluggish."

In October, 1907, she "had a dizzy turn. Had been sitting in the sun, and then while walking toward the house she sank down, but did not become unconscious." A few days later she had a similar turn and fell and hurt her head. Dr. McPhedran saw her with me, and we sent her into a private hospital for complete rest and observation. For about a month after entering here she seemed about the same, frequently complaining of headache and suffering from severe constipation, which drugs seemed almost powerless to relieve. Upon November 3, 1907, she seemed drowsy, although bright and cheerful when roused. The next day there was some diarrhœa—a most uncommon symptom for her—and later in the day she vomited. The next day the following note was made: "She vomited once during the night. No more diarrhœa. Since this morning she seems drowsy and now (4 P.M.), can scarcely be roused. Seems as if intoxicated, and if addressed smiles feebly, but will not put out her tongue or turn in bed. Resists arms being moved. There is retention of urine, and this, on being drawn off with a catheter, shows no abnormality. There is no squint and the pupils are normal." Late that evening Dr. J. M. MacCallum examined the eyes and reported that they were practically normal, the veins being perhaps a little full, and slight haziness of the left fundus. By now she was absolutely comatose, with loud stertorous breathing, and fixed pupils, and it seemed as if she must die before morning. She was evidently suffering from acute cerebral compression of some sort. Next day, however, she was no worse. A lumbar puncture was done, and the fluid seemed under some increased pressure. The cerebrospinal fluid showed no chemical or physical abnormality upon most careful examination. No choline was present.

November 7. "She is rather brighter, and feels tickling of the soles of the feet. Pupils are unequal, the left being large and the right small. They do not react to light. Marked ankle clonus present in right foot. Both knee-jerks are exaggerated. A spastic athetoic condition is present in the whole body, but especially on the left side."

November 8: "She was markedly better, being quite intelligent, and recognizes people. Answers questions quickly and says that her tea 'is hot and too sweet.' The spastic condition of the muscles is gone, and the plantar reflexes are flexor in type. Pupils are equal and react normally to light. She still requires the catheter."

On December 9 it was noted that "she is quite bright and speaks better than she has done for months. No headache." She steadily improved, and on December 13 remarked that "it was a pleasure to eat and to sleep." There was no headache. Bowels were moving naturally, which they had not done for years. Urination was normal and the reflexes were also normal.

December 24, 1907. "Doing well, but there is some want of coördination in the movements of the right hand and arm. The grip is strong, but when she feeds herself she is apt to spill things, and therefore tends to use chiefly the left hand. Is easily tired."

In January she had a similar attack of what one might call cerebral compression, in which she became very dull and difficult to rouse. The right side of the body was distinctly weak, and the right pupil was larger than the left. There was slight ankle clonus on the right side and marked on the left. She had some vomiting. She got over this attack more easily than the first one, and on February 12 returned home apparently as well as when she entered the hospital three months before.

On February 15 it was noted that "she seemed well last night and slept well until 4 A.M., then became restless and soon vomited her evening meal unchanged. Vomited twice more during the morning, became drowsy, and by 3 P.M. was quite unconscious, lying on the back with head turned toward the left and the limbs somewhat spastic. Face pale, temperature normal, and pulse sixty. Pupils small, equal, and react to light. Ankle clonus marked in *left* leg and slightly present in right. Planter reflex active and extensor in type on both sides." By 10 P.M. the pulse was 108, temperature 99.5°, and the systolic blood pressure was 210 mm. (it had frequently been measured between the attacks and never was found to be above 125 mm.). During the whole of next day she seemed to be at death's door with acute cerebral compression; but the next morning it was noted that the blood pressure had fallen to 120 mm., and by evening she was slightly conscious. The next day she was back to a condition normal for her between the attacks.

From then until March 14 she seemed to improve and was getting about the house; reflexes, pupils, etc., remained normal. The blood pressure on frequent measurements never rose above 130 mm. On March 14, 1907, she "woke at 5 A.M. feeling nauseated, and had some headache. Was dull all day, but the blood pressure did not rise above 120 mm., and by 10 P.M. she was feeling better and was sitting up in bed." Next day she seemed as usual, except that there was slight ankle clonus on the left side, and the planter reflexes were both extensor in type.

On April 8 this note occurs: "Was particularly well yesterday and walked downstairs and was speaking well. At midnight seemed restless, and then slept badly, and passed water involuntarily. This morning she seems dull, but there has been no vomiting. Is quite intelligent and answers questions clearly. There is some spasticity, best marked upon the right side. Ankle clonus and Babinski's sign well marked upon both sides." By night she was "much worse, and the spasticity almost amounts to convulsions, passing over the body in long waves. Systolic blood pressure 165 mm., but is hard to measure owing to the convulsive waves."

April 9, 9 A.M. "Had a bad night and is now quite unconscious. Spasticity very marked, but not in waves like yesterday. Pupils equal and small. Urine involuntary. Systolic blood pressure 150 mm."

From that time on the coma deepened, and the breathing got slower and slower, and she became livid. The blood pressure fell to 120 mm., but without the usual cerebral improvement. The pupils became very unequal, the left being large and the right pin-point, and later they became equally dilated. The breathing became slower and slower and then stopped altogether, the heart continuing to beat for several minutes after the last breath.

The report of the postmortem examination performed some sixteen hours after death by Professor J. J. Mackenzie is as follows:

On removing the clavarium the dura mater was found to be tense. The sinuses were filled with soft clot. On stripping the dura from the cerebrum it was found to be adherent over a large tumour mass which lay on and in the left cerebral hemisphere (Figs. 1 and 2). There were a few small flat white tumor nodules on the inner side of the posterior portion of the dura over the left hemisphere and falx cerebri. There was a very large tumor situated between the tentorium and the occipital lobe on the left side; this was attached firmly to the tentorium and easily lifted away from the brain substance. There was considerable œdema of the pia-arachnoid; the vessels of the left side were markedly congested and the convolutions were flattened. There was some dilatation of the lateral ventricles.

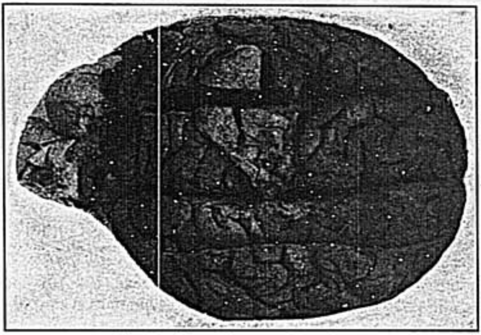

FIG. 1.—View of the tumor from above.

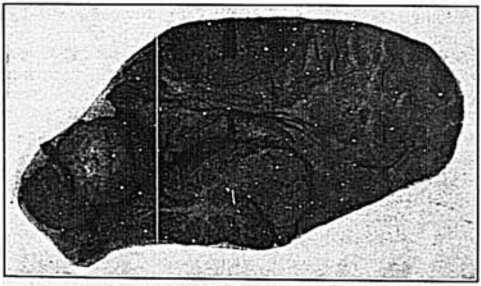

FIG. 2.—Section of the tumor.

The large tumor attached to the tentorium is roughly egg-shaped in outline and measures 6 cm. in its long axis. The larger end is 4.3 cm. in diameter; the smaller, 4.3 cm. in diameter. On section the tumor mass is very hard and fibrous, with a glistening cut surface. It lies upon and is attached to the tentorium on the left side, compressing and pushing upward the occipital lobes, compressing especially the gyrus lingualis, gyrus cuneus, and a portion of the lateral occipital gyri. The smaller tumor masses are attached to the dura and the falx cerebri, the largest measuring 0.7 cm. in diameter, all of them quite flat, 1 to 2 mm. in thickness. They do not in any way compress the brain substance, are white in color, and of the same consistency as the large mass which is attached to the tentorium. The second large tumor, which lies upon the left hemisphere, is firmly attached to the dura. It does not infiltrate the brain, but compresses the convolutions beneath it. It is loosely adherent to the pia-arachnoid, but can be dissected off. It measures 5 cm. in its anteroposterior diameter, 4 cm. lateral diameter, and is 2 cm. in thickness. It is quite soft and of a pinkish color, with here and there areas of a deeper hemorrhagic tint, and showing throughout its substance yellow areas of fatty degeneration. In front it lies upon the gyrus centralis posterior, compressing it and pushing it forward and to the left. It also compresses most of the lobulus parietalis superior, and the lobulus parietalis inferior, which appears as a narrow band between the tumor and the marginal gyrus. The inner margin of the tumor is 2.2 cm. from the longitudinal fissure. On section the gray and white matter of the brain beneath the tumor, although distorted by the compression, do not show any gross evidence of degeneration.

HISTOLOGY. The large hard tumor attached to the tentorium is exceedingly fibrous. Stained by the van Giessen method, it shows masses of red staining fibres. Between them are many cells with dark rod-like nuclei mixed with a small number of cells with large oval vesicular nuclei and faintly staining chromatin. In some areas these latter cells are present in larger numbers. The connective tissue fibers run in bundles in all directions. The vessels are well formed and are not numerous. Corpora amylacea are present but are not numerous. Mitotic figures are not seen. The soft tumor upon the surface of the parietal lobe is very cellular. The cells are somewhat smaller than the large cells of the other tumor. They are in the form of short spindles with oval vesicular nuclei. They are arranged in strands and bundles which are sometimes definitely related to the bloodvessels, and sometimes seem to run independently of them. There is very little fibre formation and the whole tumor is distinctly loose in texture. The vessels are numerous and thin walled. Mitoses are rare in this tumor also. Occasionally one finds multinucleated cells.

The histological characters of these tumors seem to point without doubt to their being endotheliomatic, arising from the dura. The large soft tumor is of the ordinary cellular type of endothelioma; the large hard tumor and the small metastatic nodules in the neighborhood would fall into the class of so-called fibro-endotheliomas. There seems no doubt that they are similar in orign, the large tumor attached to the tentorium being probably the oldest, the soft tumor over the parietal region being the most recent. In connection with the clinical symptoms the loose texture of this latter nodule and the rich vascular supply is of great significance.

Resume. The patient for the greater part of her life had suffered from severe headaches, with some aphasia during the paroxysms. At the menopause the headaches rather lessened in acuteness, but the aphasia tended to last slightly, so that she would hesitate in speech and occasionally use the wrong words, both in speaking and writing. Next, she got an attack of what seemed like acute cerebral compression without localizing symptoms. This passed off, and she was as well as before for nearly ten weeks. Then the compression recurred, and again passed off, but recurred at intervals of a month, until the fifth proved fatal. Between the attacks she would seem nearly well, and on the day before the fatal one, spoke unusually well and walked down stairs.

The case during life seemed to us to be probably one of some sort of cyst, which periodically filled and after producing almost fatal compression emptied itself in some way. The postmortem findings were a surprise, and even yet it is difficult to explain the case. The spasticity from which she suffered during the acute attacks was nearly always most marked upon the left side, that is on the same side as the tumors were. This was probably due to cerebellar irritation from the pressure of the posterior tumor. The almost constant monthly recurrence of the acute compression is interesting. It suggests the possibility of the increased pressure being in some way connected with the menstrual function, although this had ceased over a year before. She always had had bad headaches at that time, and they continued after the flow had ceased, and the same cerebral congestion now occurring in a brain the seat of tumors had in some way produced the severe and finally fatal compression. It seems, then, that the case was one of almost symptomless brain tumor, complicated by periodic attacks of cerebral congestion, this congestion being a relic of the menstrual periods.

Vasomotor disturbances are very common at the menstrual epoch, and often persist after the menopause, as in a case recently come across in which a woman had suffered most of her life from epistaxis during the menstrual period, and after the menopause this bleeding persisted and was still occurring at the age of sixty.

The blood pressure always was very high during an acute attack. We always assumed that this was due to the cerebral compression, but Professor Adami suggested that the increased blood pressure might have been due to some unexplained cause, but very likely related to the menstrual period, and that, by producing great distention of the vascular tumors, might have been the cause of the acute cerebral compression. The anterior tumor being very vascular might well lend itself to such distention.